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Aspergillus* triggers phenazine production in *Pseudomonas aeruginosa

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Objectives:

Pseudomonas aeruginosa is an opportunistic human pathogen, commonly infecting cystic fibrosis (CF) patients. Aspergilli, especially *Aspergillus fumigatus*, are also frequently isolated from CF patients. Our aim was to examine the possible interaction between *P. aeruginosa* and different *Aspergillus* species.

Methods:

A suspension of fungal spores was streaked onto WATM agar plates. After 24 hours incubation at 37  C, a *P. aeruginosa* overnight culture was streaked out perpendicular to the fungal streak. The plates were incubated at 37  C for five days, examined and plugs were extracted for HPLC-DAD and HPLC-DAD-MS analysis.

Results:

P. aeruginosa PAO1 suppressed growth of *A. fumigatus*, *A. niger*, *A. flavus*, *A. oryzae*, *A. terreus* and *Emmericella nidulans*. HPLC and HPLC-DAD-MS results showed an increase in phenazine-1-carboxylic acid and phenazine-1-carboxamide production by *P. aeruginosa* in the contact area of *A. niger*, *A. flavus*, *A. oryzae*, but not *A. fumigatus*. In addition, other metabolites with UV chromophores similar to the phenazines were only found in the contact zone between *Aspergillus* and *Pseudomonas*. No change in secondary metabolite profiles were seen for the Aspergilli, when comparing with or without the presence of *Pseudomonas*.

Conclusion:

All Aspergilli tested, with the exception of *A. fumigatus*, triggered the upregulation of phenazine-1-carboxamide and phenazine-1-carboxylic acid production by *P. aeruginosa*. Surprisingly no changes in secondary metabolite profiles were detected in any of the Aspergilli.